ORIGINAL COMMUNICATION

J. Kalita
U. K. Misra

Somatosensory evoked potential studies in internal capsule and corona radiata infarction

J. Kalita · U. K. Misra (✉)
Department of Neurology,
Sanjay Gandhi Postgraduate Institute
of Medical Sciences, Lucknow, India
e-mail: ukmisra@sgpgi.ren.nic.in
Fax: 91-0522-440973

Abstract To document the somatosensory evoked potential (SEP) changes in capsular and corona radiata infarction and correlate these with clinical and radiological findings, 15 patients with corona radiata and 16 with internal capsular infarction were studied. The mean age of the patients was 55 years (range 26–80), and 6 of them were female. In the patients with corona radiata infarction, median N9–N20 conduction time was abnormal in 4 cases, which correlated with sensory abnormalities in 1. In 3 of these patients, infarction was located in the anterior two-thirds and in 1 there was total corona radiata infarction. The amplitude of N20 potential on the affected side was reduced in 1 patient. In the capsular infarction group, N9–N20 conduction time was abnormal in 1 patient only who had total involvement of the posterior limb of the internal capsule. The amplitude of N20 was reduced in another patient. There were 4 patients who had abnormal sensory findings, but their SEPs were normal. At 3 months, the SEP changes remained stable in all of the patients who were followed up. The SEP changes did not correlate with changes in sensation or 3-month outcome as assessed by the Barthel index score. The lack of clinicoradiological and SEP correlation may be owing to variation on the organisation of sensory pathways in the corona radiata and internal capsule.

Key words Infarction · Corona radiata · Internal capsule · Somatosensory evoked potential

Introduction

The organisation of sensory pathways, especially from the thalamus to the cerebral cortex has fascinated neuroscientists for a long time. Anatomical and stimulation studies have suggested that both motor and sensory pathways are organised somatotopically all along their courses. In the cerebral cortex, the sensory and motor pathways are organised in the form of a homonculous [23]. The somatotopic organisation is not only maintained in the cerebral cortex but also in the internal capsule [11]. Clinical and radiological correlation studies have, however, reported striking deviation from the expected pattern of the motor pathways in the internal capsule [21]. Studies of clinical, radiological and motor evoked potential (MEP) changes in capsular and corona radiata infarction have revealed that clinical and MEP findings in the internal capsular infarction follow a more predictable pattern compared with the corona radiata infarction [9, 10, 15]. For diagnosing and documenting the sensory abnormalities, somatosensory evoked potentials (SEPs) are invaluable and have been extensively studied in patients with ischaemic stroke [13, 18, 24–26]. There have been a large number of studies on SEPs following stroke [15, 17]. Most of these have investigated SEP changes in different locations of stroke. Follow-up studies correlating clinical and evoked potential changes have been reported in only a few studies [15, 17]. SEP studies can be useful for studying the organisation of sensory pathways in radiologically defined dis-

crete subcortical regions affecting the sensory pathways. In the available literature, we could not find such a study. We now report the median SEP changes and their clinico-radiological correlation in patients with corona radiata and capsular stroke and their role in predicting the 3-month outcome.

Materials and methods

The patients with corona radiata and internal capsule infarction have been included in the study from a group of consecutive stroke patients managed by us from 1991 to 1996. The patients were examined within 2 weeks of ictus, and the diagnosis was confirmed by computed tomography (CT). Cranial CT was carried out in all patients within 1 week of ictus employing third generation CT scanner W400 Hitachi, Japan. CT was carried out parallel to the orbitomeatal line, obtaining 10-mm sections. The corona radiata infarctions were classified into anterior, middle and posterior third and internal capsular infarctions into anterior limb, genu and pos-

Internal capsular infarctions

Corona radiata infarctions

Fig. 1 Schematic diagram of classification of internal capsular (*upper panel*) and corona radiata (*lower panel*) infarctions. Capsular infarctions were classified into *A* = anterior limb, *B* = genu and *C* = posterior limb. The latter was further subdivided into anterior, middle and posterior third. The corona radiata infarctions were classified into *a* = anterior third, *b* = middle third and *c* = posterior third. The numbers represent the cases of particular subtype

terior limb. Posterior limb infarctions were further subdivided into anterior middle and posterior third (Fig. 1).

Median SEPs were obtained by stimulating the median nerve at the wrist. Each side was stimulated separately. The patients were asked to relax in the supine position. Active recording surface electrodes were placed at the Erb's point and contralateral parietal cortex (7 cm lateral and 3 cm behind Cz, CPC). The reference electrodes were placed at Fz for both Erb's point and CPC recordings. The electrode impedance was kept below 5 kΩ. The median nerve was stimulated by 0.5- to 1-ms square wave pulses at 1 Hz ensuring a painless twitch of the thumb. The overall handpass was 2–3000 Hz and the analysis time 100 ms; 512 responses were averaged twice to ensure reproducibility. The N20, P25, N30, P45 waveforms were measured in CPC-Fz channel and N9 potential in Erb's point-Fz channel. the normal values were determined using the same technique, for an age- and sex-matched population of 32 normal volunteers. The normal values were defined by mean ± 2.5 SD of controls [19]. The upper limit of N9–N20 conduction time in the control group was 11.3 ms (8.3, SD 1.2). The upper limit of N20-P45 conduction time was 32.3 ms (22.3, SD 4.0). The amplitude of N20 was 2.8, SD 2.2 μV and that of P45 was 3.9, SD 2.8 μV. The amplitude of N20 and P45 have shown a wide variation in the normal population, therefore the amplitude of the unaffected side was used for comparison. A reduction of 50% or more was considered abnormal [6].

The SEP studies were repeated at 1 and 3 months. The relationship between clinical, radiological and SEP changes was evaluated by the χ^2 test. For defining abnormality of median SEP, both the latency (N9–N20) and the amplitude of N20 have been considered. The P45 amplitude and P45–N20 conduction time, although measured, have not been used in defining abnormality.

Results

Our results are based on 31 patients with internal capsule and corona radiata infarctions. All of the patients were right-handed, their mean age was 55 years (range 26–80) and 6 of them were female. The patients were examined after a mean duration of 6 days (range 1–15). Fifteen patients had corona radiata and 16 internal capsule infarctions.

Internal capsular infarction

There were 16 patients with capsular infarction; the mean age of these patients was 54 years (range 26–70) and 3 of them were female. These patients were examined after a mean duration of 5.9 days (range 1–14). Impairment of pinprick and joint position sensation was present in 4 patients, which returned to normal in 3 at 3-month follow-up. The infarctions were located in the anterior limb in 1 patient, in the genu in 4, in the genu with anterior third of the posterior limb in 5 and isolated anterior third and posterior third of the posterior limb in 1 each, and in the middle two third of the posterior limb in 2. Complete posterior limb infarction was found in 2 patients. The N9–N20 conduction time of median SEP was abnormal in 1 patient only who had complete posterior limb infarction. Interestingly, the clinical sensory testing in this patient was normal. In the 4 patients with abnormal sensory findings, the
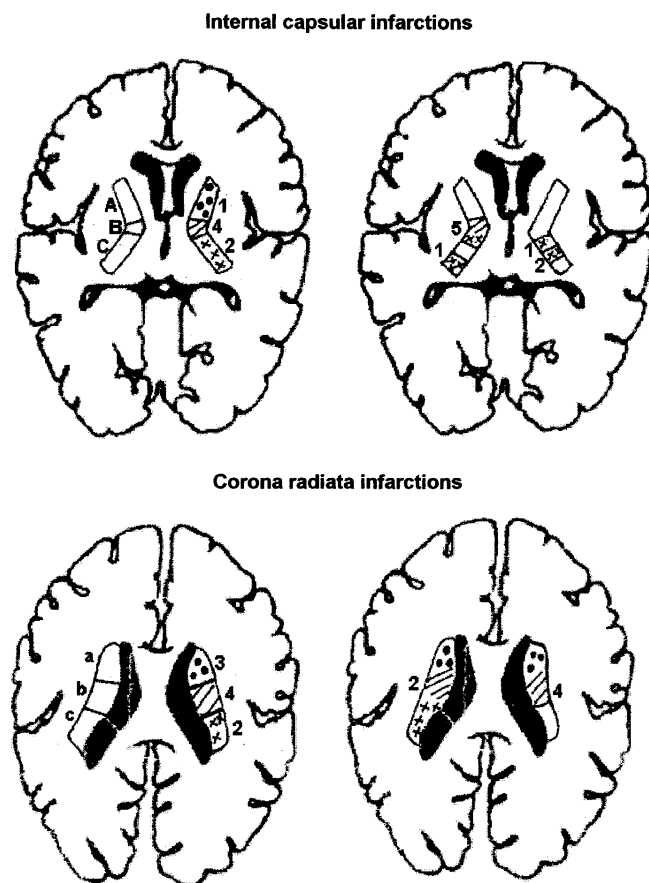
Table 1 Sensory functions in patients with internal capsule infarction (*PP* pinprick, *JPS* joint position sense, *C* cortical, *N* normal, ↓ reduced, *NR* not recordable, *PL* posterior limb, *Ant* anterior limb, *CSCT* central sensory conduction time, *Mid* middle)

No.	Age/sex	Day	Sensory function			CSCT		Location	Outcome
			PP	JPS	C	N9–N20	P45-N20		
1	45F	1	↓	↓	N	10.2	27.0	Ant	Complete
		90	N	N	N	10.3	27.2		
2	56M	2	↓	↓	N	10.0	22.0	Genu	–
3	54M	4	N	N	N	9.2	23.3	Genu	Complete
		90	N	N	N	9.1	23.6		
4	53M	12	N	N	N	10.0	23.6	Genu	–
5	43M	2	N	N	N	8.6	27.2	Genu	Complete
		90	N	N	N	8.6	27.0		
6	52M	3	N	N	N	9.2	17.6	Genu & Ant 1/3 PL	Partial
		90	N	N	N	9.3	17.5		
7	55M	12	N	N	N	10.2	27.6	Genu & Ant 1/3 PL	Partial
		90	N	N	N	10.0	27.5		
8	26M	14	↓	↓	N	9.4	19.6	Genu & Ant 1/3 PL	Complete
		90	N	N	N	9.2	19.8		
9	62M	3	N	N	N	10.0	27.8	Genu & Ant 1/3 PL	Partial
		90	N	N	N	10.2	27.4		
10	57M	8	↓	↓	N	8.6	19.6	Genu & Ant 1/3 PL	Complete
		90	N	N	N	8.4	19.0		
11	65M	6	N	N	N	NR	NR	PL	Poor
		90	N	N	N	NR	NR		
12	70M	2	N	N	N	10.0	16.0	PL	Complete
		90	N	N	N	10.0	18.0		
13	70F	3	N	N	N	9.6	32.8	Ant 1/3 PL	Partial
		90	N	N	N	9.6	31.8		
14	65M	14	N	N	N	10.0	25.0	Post 2/3 PL	–
15	35M	3	N	N	N	9.2	28.0	Mid 2/3 PL	–
16	58F	5	N	N	N	10.0	NR	Mid 1/3 PL	Partial
		90	N	N	N	9.2	19.6		

infarctions were located in the anterior limb in 1, in the genu in 1 and in the genu with the anterior third of the posterior limb in 2 patients. The P45 waveform was unrecordable in 2 and the P45–N20 conduction time was prolonged in 1 patient. These patients had normal sensation. The amplitude of N20 on the affected side ranged between 0.9 µV and 7.8 µV (mean 2.46) and on the normal side between 1.0 µV and 7.6 µV (mean 2.6). Only 1 patient had more than 50% amplitude reduction on the affected side (patient 14). In this patient, the N9–N20 conduction time and clinical sensory testing were normal, and the infarction was located in the posterior third of the posterior limb of the internal capsule. The amplitude of P45 on the affected side ranged between 1:1 µV and 10.6 µV (mean 4.79) and on the normal side between 0.8 µV and 9.0 µV (mean 3.86). None of the patients with capsular infarctions had a significant reduction in P45 amplitude.

The clinical, radiological and SEP findings in capsular infarction are summarised in Table 1.

Corona radiata infarction

There were 15 patients with corona radiata infarction. The mean age of the patients with corona radiata infarction was 56 years (35–80) and 3 of them were female. The patients were examined after an average of 6 days of ictus (range 1–15). Sensory abnormalities were present in 1 patient who had impaired pinprick and joint position sensation. In 1 patient, sensory testing was not reliable because of aphasia (patient 14). The middle third of the corona radiata was involved in 4, the anterior third in 3, the posterior third and total corona radiata were involved in 2 patients each and the anterior two-thirds was involved in

Fig. 2 Median SEP in a patient with corona radiata infarction located in the two-thirds (patient 11) with prolonged N9–N20 conduction time (12.8 ms). This patients had normal sensation
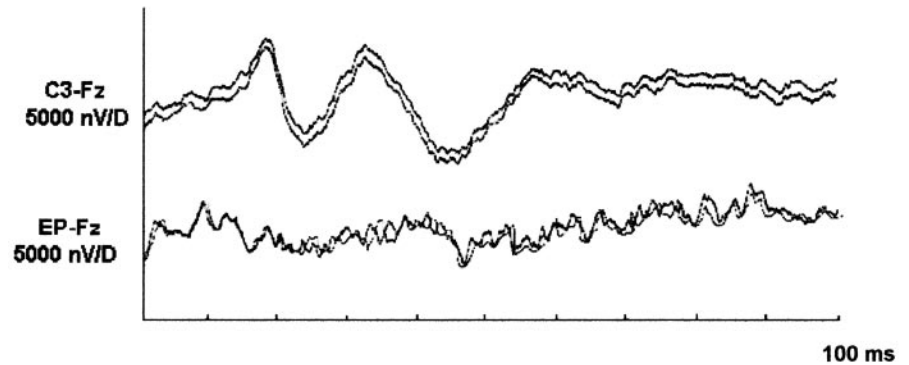

C3-Fz
5000 nV/D

EP-Fz
5000 nV/D

100 ms

Table 2 Sensory functions in patients with corona radiata infarction (*Ant* anterior, *Post* posterior)

No.	Age/sex	Day	Sensory function			CSCT		Location	Outcome
			PP	JPS	C	N9–N20	P45-N20		
1	50M	1	N	N	N	10.4	25.0	Mid 1/3	Poor
		90	N	N	N	11.2	29.2		
2	80M	2	N	N	N	10.0	22.4	Mid 1/3	Partial
		90	N	N	N	10.0	22.8		
3	42M	2	N	N	N	8.2	NR	Mid 1/3	–
4	45F	5	N	N	N	8.0	18.4	Mid 1/3	Complete
		90	N	N	N	8.0	18.6		
5	52F	3	N	N	N	8.8	30.2	Ant 1/3	Complete
		90	N	N	N	8.8	30.2		
6	62F	7	N	N	N	10.6	15.2	Ant 1/3	–
7	60M	5	N	N	N	9.0	20.0	Ant 1/3	–
8	48M	8	N	N	N	9.6	23.2	Post 1/3	Complete
		90	N	N	N	9.6	23.0		
9	60M	4	N	N	N	NR	NR	Ant 2/3	Poor
		90	N	N	N	10.4	26.0		
10	78M	4	↓	↓	–	NR	NR	Ant 2/3	Poor
		90	↓	↓	–	NR	NR		
11	70M	9	N	N	N	12.8	33.2	Ant 2/3	–
12	35M	9	N	N	N	10.4	NR	Ant 2/3	Poor
		90	N	N	N	10.2	NR		
13	40M	15	N	N	N	8.4	20.2	Post 1/3	–
14	63M	14	?	?	?	NR	NR	Total	–
15	55M	4	N	N	N	9.2	25.2	Total	Partial
		90	N	N	N	10.0	21.4		

4 patients. Median central sensory conduction time was normal in 4 patients, unrecordable in 3, and there was a prolonged N9–N20 conduction time in 1 (Fig. 2). In these patients, only 1 had abnormal sensation on clinical examination (patient 10). The location of infarction in these patients was in the anterior two-thirds of the corona radiata in 3 (patients 9–11) and total corona radiata in 1 (patient 14). Two of these patients were followed up for 3 months and both had poor recovery. On correlating the central sensory conduction time with radiological findings, it was found that SEPs were normal in isolated anterior, middle and posterior third infarctions and even in complete corona radiata infarction; however, SEP changes were more frequent in patients with anterior two-thirds corona radiata infarction (3 out of 4 patients). The amplitude of N20 on the affected side ranged between 0.8 µV and 3.5 µV (mean 1.9) and on the normal side between 0.5 µV and 4.8 µV (mean 1.9). The amplitude of N20 potential was

significantly reduced on the affected side in 1 patient only (patient 12) although his N9–N20 conduction time and sensations were normal. The location of infarction in this patient was in the anterior two-thirds of the corona radiata. The amplitude of P45 on the affected side ranged between 0.8 μV and 10.8 μV (mean 3.6) and on the normal side 1.1 μV and 10.2 μV (mean 3.5). Only 2 patients had significant reduction of P45 amplitude on the affected side (patients 4, 15). Both of these patients had normal sensation and N20–P45 conduction time. The results of sensory testing, SEP studies and CT findings in the patients with corona radiata infarction are presented in Table 2.

Follow-up and outcome

Nine patients with corona radiata and 12 with capsular infarction were followed up. At 3 months, the sensory impairment improved in 3 patients with capsular infarctions. In another patient with capsular infarction, the SEP remained unrecordable even at 3 months. In patients with corona radiata infarction, the abnormal sensory testing as well as SEP abnormaliti persisted in 1. In another patient, however, unrecordable SEP became normal at 3-month follow-up. The outcome in the patients with corona radiata and capsular infarctions did not correlate with sensory and SEP abnormalities. In the patients with corona radiata infarctions, 3 had complete, 2 partial and 4 poor recovery. In the capsular infarction group, 6 patients had complete, 5 partial and 1 poor recovery. With capsular infarctions, sensory abnormality ($\chi^2 = 1.8$, $df = 1$, NS), SEP abnormality ($\chi^2 = 2.2$, $df = 1$, NS), and in the corona radiata group the sensory abnormality ($\chi^2 = 0.1$, $df = 1$, NS) and SEP abnormality ($\chi^2 = 0.1$, $df = 1$, NS) were not significantly related to the outcome.

Discussion

In our study, sensory abnormalities on clinical testing were present in 5 patients, 1 with corona radiata and 4 with capsular infarction. The SEP, however, was abnormal (N9–N20 conduction time and N20 amplitude) in 5 patients (16%); 4 patients with corona radiata and 2 with capsular infarction. The SEP abnormality correlated with clinical sensory loss in only 1 patient with corona radiata infarction in our study. A better correlation between clinical sensory testing and SEPs has been reported in a number of earlier studies [18]. In a study of MEP and SEP, out of 6 patients with lacunar infarction, the N20 potential was unrecordable in the patients with sensory deficit [17]. In lacunar infarction, SEP was abnormal in 30% of patients [2]. In another study on cerebral infarction (4 cases of striatocapsular infarction, 4 cases of lacunar infarction and 1 case of subcortical infarction) sensory abnormalities were present in 6, whereas SEP was abnormal in 5 of these patients. In 3 patients, however, the abnormalities were in the waveforms beyond N20. A higher frequency of SEP abnormalities in other studies may be attributed to the inclusion of waves beyond N20 [17], the amplitude criteria [15, 17] and the inclusion of larger infarction [15, 17]. The long latency cortical potentials are variable even in normal subjects [4, 5]. In the younger subject, the median SEP may have a V pattern instead of a W pattern, which is common in old age [9]. We have therefore not included the waveform beyond N20 in statistical analysis, although these were measured.

Sequential study of SEPs in our patients revealed that these were rather stable; in one patient only, unrecordable SEP became normally recordable at 3 months. A high frequency of SEP abnormality (48%) of cerebral infarction has been reported in the acute stage, which declined to 39% at 2–3 months and to 29% at 1 year follow-up [15]. In another study, in most patients SEP either remained normal or tended to become normal in the follow-up period [17]. In our earlier studies SEP changes in putaminal haemorrhage were also stable [20], as they were in thalamic haemorrhage with posterolateral extension [19]. The change in SEP following ischaemic stroke is attributed to recovery of neuronal function following perfusion being restored in the ischaemic area. This may occur from a number of processes such as breaking up of emboli, restoring the patency of previously occluded vessels or opening of collateral channels and resolution of oedema, thus providing a return of cerebral function [5, 22]. SEPs have been reported to continue to improve even after 6 months of stroke in some patients [17]. We however, did not follow our patients after 3 months; therefore the later improvements in SEP cannot be commented on the basis of our results.

The N20 potential of median SEP is regarded as originating in the primary sensory cortex [23], the thalamus [1, 7], the thalamocortical projections [8] and the parietal cortex [12]. Most of the recent reports, however, favour a cortical origin of SEPs. A study employing cortical surface and transcortical depth recording during epilepsy surgery revealed the origin of N20 and P30 potentials in area 3b, in the posterior lip of the central sulcus. Similar recordings were obtained from subcortical areas of the frontal region, which persisted even after removal of the hand area of the motor cortex [4]. On median nerve stimulation, there was a single focus in the primary sensory area, which linearly correlated with regional cerebral blood flow measured by positron emission tomography (PET). The supplementary motor area during median nerve stimulation was neither significantly activated nor was there any increase in blood flow. This study raises doubts about the contribution of the primary motor or supplementary motor cortex to SEP waveforms [14]. In our study, therefore, we measured the N20 potential and did not study the frontal generator N30 in Fz-EPc (contralateral Erb's point) derivation.

The patient with capsular infarction having genu and anterior limb involvement had a better outcome compared with those with total posterior limb infarction. Corona radiata infarction involving two-thirds or more resulted in a poor outcome compared with a smaller infarction. This generalisation did have exceptions; a patient with complete corona radiata infarction had a normal SEP (patient 15). The extent of infarction did not follow the correlation with sensory loss or SEP abnormalities. This could be owing to the diffuse organisation of sensory pathways and the escape of some fibres resulting in normal SEP. Using N20 amplitude and N9–N20 conduction time as abnormality criteria, the SEPs are abnormal in only 16% of patients with capsular and corona radiata infarctions. The lesion has to be extensive, involving the whole of the posterior limb of the internal capsule or two-thirds of the corona radiata, to result in a poor outcome. It can be concluded from our study that SEP abnormalities do not follow a predictable pattern following capsular or corona radiata infarction, which may be owing to variation in the organisation of sensory pathways.

References

1. Abbruzzese M, Favale E, Leandri M, Ratto S (1978) New subcortical components of cerebral somatosensory evoked potentials in man. Acta Neurol Scand 58:325–332
2. Abbruzzese G, Bino G, Dall-Agata D, Primavera A, Favale E (1988) Somatosensory evoked potentials lacunar syndromes. J Neurol 235:300–303
3. Allison T, McCarthy G, Wood CC, Darcey DD, Spencer DD, Williamson PD (1989) Human cortical potentials evoked by stimulation of median nerve. Cytoarchitectonic areas generating short latency activity. J Neurophysiol 62:694–710
4. Allison T, McCarthy G, Wood CC, Williamson PD, Spencer DD (1989) Human cortical potentials evoked by stimulation of median nerve. (II) Cytoarchitectonic areas generating long latency activity. J Neurophysiol 62:711–722
5. Bell BA, Symon L, Branston NM (1985) CBF and time thresholds for the formation of ischaemic cerebral oedema and the effect of reperfusion in baboon. J Neurosurg 62:31–41
6. Chester CS, McLaren CE (1989) Somatosensory evoked response and recovery from stroke. Arch Phys Med Rehabil 70:520–525
7. Chiappa KH, Choi S, Young RR (1980) Short latency somatosensory evoked potentials following median nerve stimulation. In: Desmedt JE (ed) Progress in clinical neurophysiology, vol 7. Karger, Basel, p 264
8. Cracco RQ, Anziska BJ, Cracco JB, et al (1992) Short latency somatosensory evoked potentials to median and peroneal nerve stimulation studies in normal subjects and patients with neurologic disease. Ann N Y Acad Sci 388: 412
9. Desmedt JD (1988) Somatosensory evoked potential. In: Picton TW (ed) Handbook of electroencephalography and clinical neurophysiology, vol 3. Elsevier, Amsterdam, pp 245–360
10. Ferbert H, Buchner H, Brucknann H, Zeumer H, Hacke W (1988) Evoked potentials in basilar artery thrombosis: correlation with clinical and angiographic findings. Electroencephalogr Clin Neurophysiol 69:136
11. Fries W, Dane KA, Scheid Hetmann K, Hamburger C (1990) Motor recovery following capsular stroke. Brain 116:369–382
12. Giblin DR (1964) Somatosensory evoked potentials in healthy subjects and in patients with lesions of nervous system. Ann N Y Acad Sci 112:94–142
13. Graff-Radford NR, Damasio H, Yamada T, Eslinger PJ, Damasio AR (1985) Nonhaemorrhagic thalamic infarction. Clinical neurophychological and electrophysiological findings in four anatomical groups defined by computerised tomography. Brain 108:485–516
14. Ibanez V, Deiber M, Sadato M, Torco C, Grisson J, Woods RP, Mazzlatta JC, Hallett M (1995) Effect of stumulation rate on cerebral blood flow on median nerve stimulation. Brain 118:1339–1351
15. Kovala T, Tolonen U, Pyhtinen J (1993) A prospective one-year follow up study with somatosensory potentials evoked by stimulation of the median nerve in patients cerebral infarcts. Electromyograph Clin Neurophysiol 33:359–367
16. Larsen SJ, Sancer A, Barker JBN (1966) Evoked cortical potentials in patients with stroke. Circulation 33 [Suppl II]:92
17. MacDonell RAL, Donnan GA, Bladin PF (1991) Serial changes in somatosensory evoked potentials following cerebral infarction. Electromyograph Clin Neurophysiol 80:276–283
18. Mauguiere F, Desmedt JE, Courjon J (1983) Asteriognosis and dissociated loss of frontal and parietal components of somatosensory evoked potential in hemispheric lesion. Brain 106:271–311
19. Misra UK, Kalita J (1996) Evoked potentials studies in thalamic haemorrhage. Clin Neurol Neurosurg 98:291–298
20. Misra UK, Kalita J (1996) Serial changes in motor and sensory evoked potentials in putaminal haemorrhage. J Neurol 243:73–78
21. Mohr JP (1982) Lacunes. Stroke 13: 3–11
22. Oslen TS, Bruhn P, Oberg RGE (1986) Cortical hypoperfusion as a possible cause of subcortical aphasia. Brain 109:393–410
23. Penfield W, Rasmussen TH (1954) The cerebral cortex of man. MacMillan, New York, p 44
24. Reisecker F, Witzmann A, Deisenhammer E (1986) Somatosensory evoked potentials (SSEPs) in various groups of cerebrovascular ischaemic disease. Electroencephalogr Clin Neurophysiol 65: 260–268
25. Stejskal L, Sobota J (1985) Somatosensory evoked potentials in patients with occlusions of cerebral arteries. Electroencephalogr Clin Neurophysiol 61:482–490
26. Stohr M, Dichgans J, Voigt K, Buettner UW (1983) The significance of somatosensory evoked potentials for localisation of unilateral lesions within the cerebral hemispheres. J Neurol Sci 61:49–63